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As mass communicable diseases have been brought under control, attention has been drawn to pathogens previously regarded as of little consequence. Chief of these is the Staphylococcus. Emphasis is placed in this symposium on the hospital as a source of staphylococcal disease, how infection is spread, and on the methods of prevention and control.

STAPHYLOCOCCAL INFECTIONS IN THE HOSPITAL AND COMMUNITY

HOSPITAL ENVIRONMENT AND STAPHYLOCOCCAL DISEASE

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THE STRENGTH of a hospital is especially the people who work there, but the place where they work has throughout history been of great importance to the fulfillment of their purpose. Although the hospital was intended to assist in the relief of distress and suffering, it has time and again served as a focus for the propagation of disease which has thwarted this purpose. This was a major reason why hospitals, prior to adoption of the principles promulgated by such leaders as Alanson,¹ Collins,¹⁴ Semmelweis,⁴² Nightingale,³⁵ Lister,²⁸ Simpson,⁴³ and Schimmelbusch⁴¹ were used mostly by the poor; the rich preferred medical care in the safer environment of their homes.^{9, 48}

Progress in hospital care can be

measured from the conditions existing in the Hotel Dieu in Paris in 1788: "There were some 1,200 beds, most of which contained from four to six patients, and also 486 beds for single patients. The larger halls contained over 800 patients crowded on pallets, or . . . heaps of straw, which were in vile condition. Acute contagious diseases were often in close relation to mild cases, vermin and filth abounded, and . . . the attendants . . . would not enter in the morning without a sponge dipped in vinegar held to their faces. Septic fevers and other contagia were the rule; the average mortality was about 20 per cent, and recovery from surgical operations was . . . a rarity."⁴⁵

Our modern hospitals bear little re-

semblance to such progenitors, but the transition has been slow and laborious, and for most hospitals it remains incomplete. Much remains to be done to approach Nightingale's precept that the hospital "shall do the sick no harm."³³ Fortunately, the task of preventing cross-infection in hospitals is much lighter today than it was in earlier times when great mortality resulted from many parasites in addition to staphylococci; when cholera and typhoid decimated hospital as well as nonhospital populations; when diphtheria and smallpox were frequent threats; when 2,944 of 7,650 infants born in one hospital died during the first fortnight, largely of neonatal tetanus²⁷; and when puerperal sepsis was the scourge of most lying-in hospitals.^{13, 42}

The major enteric diseases have largely yielded to improved community sanitation; immunization has virtually conquered diphtheria, smallpox, and tetanus; and streptococcal disease has been much subdued by the antibiotics. What remains now is especially staphylococcal disease, an insidious and frequently overlooked disease, but one which has survived the onslaught of community sanitation, immunization, and the antibiotics, to emerge from our hospitals as probably the foremost parasitic cause of death in many modern communities.

History and Epidemiology of Staphylococcal Disease

With few exceptions the literature dealing with staphylococcal disease is fragmented according to anatomic site, type of manifestation, and age of patient, making it difficult to estimate total staphylococcal disease incidence for a complete population in a circumscribed area during a given period of time. We will, therefore, consider the history of several major manifestations of staphylococcal disease individually.

Suppuration, pyemia, and septicemia

were the common sequelae of surgery for centuries. So common was the suppuration following surgery that, if delayed in its appearance, active measures were employed to hasten the formation of "laudable pus."⁵ But that suppuration was neither a necessary nor desirable product of surgery was demonstrated repeatedly before Pasteur provided the bacteriologic key to the puzzle. As early as 1782, Alanson¹ performed 35 amputations in the Birmingham Infirmary without loss of a single patient or a serious wound infection. He recognized rather fully the value of isolating surgical patients in a sanitary environment. Analysis of military surgical experience during the nineteenth century wars, as summarized by Cowles¹⁵ and Erichsen,¹⁸ showed that "the danger of pyemia increased . . . in proportion as the hygiene was faulty and as the wounded patients were closely crowded." Improved hospital records of the results of patient care permitted Sir James Simpson in 1869⁴³ to collect data which related the outcome of surgical treatment in Britain especially to the size, location, and condition of hospitals. He found that the mortality rate from limb amputation was four times greater in large metropolitan hospitals than in private country practice. In 1879 the Pathological Society of London³ investigated "The Nature and Causes of Those Infective Diseases Known as Pyaemia, Septicaemia, and Purulent Infection," and demonstrated that these diseases were caused by micrococci and that they usually originated in the hospitals. Table 1 shows that during a 10-year period 40 per cent of the 1,319 erysipelas deaths and 80 per cent of 261 pyemia deaths which occurred in three London hospitals originated in them. Then, as now, hospitals were the dominant source of fatal pyemia, most of which was probably caused by staphylococci.^{3, 50} With the adoption of Lister's principle of antiseptic surgery²⁸ and later of the

**Table 1—Deaths from Pyemia and Erysipelas in Three* London Hospitals:
The Relative Importance of Hospital-Acquired Infection†**

	Years										Total
	1869	1870	1871	1872	1873	1874	1875	1876	1877	1878	
Pyemia admitted to hospital	5	5	5	6	3	7	8	8	3	2	52 (20%)
Pyemia originating in hospital	13	14	22	28	5	23	20	17	27	20	209 (80%)
Erysipelas admitted to hospital	89	106	76	220	219	177‡	174‡	112	88‡	58‡	1,319‡ (60%)
Erysipelas originating in hospital	95	74	54	98	111	170	107	67	61‡	30‡	867‡ (40%)

* Middlesex, St. Thomas's, and St. Bartholomew's Hospitals.

† Constructed from data presented in reference 3.

‡ Figure less than true because of no report from a hospital.

aseptic principle and improved technics of von Bergmann, Schimmelbusch, and Halsted, the mortality from surgical wound infections diminished.⁴⁶ However, scattered reports during the twentieth century indicate that surgical wound infection by no means ceased. In 1925, Meleney³³ began keeping careful records of postoperative wound infections in a New York hospital, and found the infection rate to be 15 per cent for clean wounds, which was seven and one-half times greater than had been estimated by the chief of one of the surgical services. In 1933, Hunt²³ in Massachusetts, detected evidence of postoperative wound infection in 10 of 28 routine operative cases, and from his own experience and the literature derived "the opinion that 10 per cent expresses very conservatively the morbidity from operative wound infection under average standards of technique and equipment at the present time." In 1938, Ives and Hirschfeld²⁵ estimated that about 5 per cent of clean operative wounds in New Haven developed signs of infection and that "well over 50%" of such infections were caused by staphylococci. In 1954, Howe²² reported that in a Boston hospital 7.2 per cent of clean surgical cases developed postoperative infection and that postoperative sepsis had increased one-half of one per cent annually the last five years. Recently, in Seattle, Wysham and Kirby⁵⁰

found that staphylococcal disease acquired in hospital was a contributing factor to the death of 12 surgical patients in a general hospital during a four-month observation period. From these reports it appears that infection of surgical wounds in hospitals, especially with staphylococci, remains a considerable cause of death. In fact, the mortality from hospital-acquired pyemia and septicemia (erysipelas excluded) in London hospitals during the 1870's³ (Table 1) was not remarkably greater than current mortality from the same conditions in some of our large teaching hospitals.⁵⁰

According to Poole and Whittle (1935),³⁶ "The earliest description of pemphigus of the newly born was by Ochene in 1773, but it was not until 1865 that Tilbury Fox drew attention to the contagious nature of the disease, though epidemics had been described by Rigby in England in 1834 and by Leseque and Trousseau in France in 1850." During the next several decades, additional epidemics of pemphigus neonatorum were reported from France, Germany, and America, and attention was drawn to the association of such epidemics with certain hospitals and midwives.^{26, 36} In the first report of an epidemic in America, Kilham (1889)²⁶ differentiated the disease from "the ordinary syphilitic pemphigus," and in 1891 Almquist² identified *Staphylococcus*

pyogenes aureus as the causative organism. In 1904, Call⁷ described "An Epidemic of Pemphigus Neonatorum" in the New England Hospital where "all the babies are kept in a nursery." She noted the occurrence of associated maternal breast abscesses, and stated that "there is absolutely no means of stopping the spread of the infection except the complete isolation of both mother and child."

A gradual increase in staphylococcal disease among newborn infants in America and Britain during the period from 1917 to 1935 was documented by Reed in 1929³⁹ and by Poole and Whittle in 1935.³⁶ In Chicago "very few nurseries were exempt,"³⁹ and in one Chicago hospital five epidemics, each of one or two-months duration, occurred between 1917 and 1928. Recent reports have further documented extensive endemic and epidemic occurrence of hospital-derived staphylococcal disease in newborn infants.^{37, 38, 49} Differences in the distribution of staphylococcal disease of infants and mothers, according to hospital and time of delivery, have been clearly demonstrated.^{37, 38} A survey of mothers and infants delivered in Seattle hospitals during October, 1956, revealed average suppurative morbidity rates of 4.3 per cent maternal mastitis, 1.0 per cent infant mastitis, and 18 per cent infant pyoderma. One hospital "with 10 per cent of total deliveries . . . produced 20 per cent of the infant pyoderma, 45 per cent of the maternal mastitis, 100 per cent of the infant mastitis, and 75 per cent of the neo-infant staphylococcal disease mortality, which occurred among 1,456 mothers and their infants who were delivered in 15 Seattle hospitals during October, 1956."³⁸ Seventy-six per cent of the mothers who delivered in this one hospital and who attempted to nurse their infants developed mastitis. The usual chain of infection has been found to be from nursery-to-infant-to-nursing mother. Sup-

port for this principle of transmission is provided by the close association, in time and place, of suppurative illness of infants and mothers; by the usual precedence of infant infection; by the predilection of maternal mastitis to occur in nursing mothers; by the usual recovery of the same type of staphylococcus from infant and maternal lesions; and by the abrupt cessation (by date of delivery) of both infant and maternal disease when effective preventive measures are instituted in the nursery. Furthermore, the literature, as reviewed above, indicates substantial correlation, historically, between the use of hospitals for childbirth and the occurrence of staphylococcal disease of newborn infants and their mothers. The nursery-infected newborn infant is an extraordinarily effective vehicle for the transmission of staphylococcal disease (and other diseases as well) to mothers and other family contacts, and to other hospital wards.

Staphylococcal pneumonia is seldom listed as the cause of death upon death certificates, yet recent studies in Seattle,^{37, 38, 50} in addition to the findings of others,^{8, 10, 16, 19, 32} indicate that staphylococcal pneumonia is a frequent cause of death, especially in surgical, debilitated, or influenza patients, and in infants. During the 1918 influenza epidemic, Chickering and Park⁸ found that in Camp Jackson, S. C., "The occurrence of *Staphylococcus aureus* in the lungs in the fatal cases was quite striking, 49 per cent, of the 312 cases cultivated showing this organism present either alone, ninety-two cases, or in association" with other bacteria. In 1949 McLetchie³² wrote that, "Workers at the Royal Hospital for Sick Children, Glasgow, record only three primary staphylococcal pneumonias in the period 1926 to 1935 in a series of 2,300 consecutive autopsies, while they record 55 cases in the period 1936 to 1945 in a series of 2,877 consecutive autopsies." Summing

up his experience in Saskatchewan, Mc-Letchie added that, "Records over the last ten years show that in Regina the *Staphylococcus* is the only organism commonly producing fatal pneumonia in infancy and childhood." The Seattle studies^{37, 38, 50} have shown that hospital-acquired staphylococcal pneumonia is a substantial cause of death among many categories of hospital patients and especially among infants during the first several months following discharge from the hospital of birth.

Reports of the now too familiar suppurative manifestations of staphylococcal infection abound throughout the medical literature. However, the first comprehensive report of widespread epidemic occurrence of such disease that we have found was by Hunt in 1852.²⁴ Hunt's description of the then unusually great world-wide prevalence of "carbuncles, boils, whitlows, pustules, and superficial collections of purulent matter" which he termed "the furunculoid epidemic" is remarkably descriptive of the current problem. Hunt presented mortality statistics for England and Wales which showed a gradual increase in deaths from "carbuncle" during the years 1840-1852. A subsequent report by Reid⁴⁰ extends the epidemic at least until 1855. The concurrence of this insidious but widespread and prolonged epidemic of what was undoubtedly staphylococcal disease, with what Colebrook¹³ has termed "the blackest period in all the story of hospital infection" is noteworthy:

For when we gather together the many manifestations of staphylococcal disease . . . the suppuration, pyemia, and septicemia following surgery; the pyoderma, conjunctivitis, gastroenteritis, mastitis, and pneumonia of newborn infants; the mastitis of nursing mothers; the pyoderma of family contacts of hospital-born infants; and more; we find that all these afflictions are branches of the

same staphylococcal disease tree . . . which has its roots in our hospitals.

The Hospital Environment

If one concludes from the evidence at hand that a large proportion of serious staphylococcal disease is currently derived from hospitals, one must ask: Why is this so?

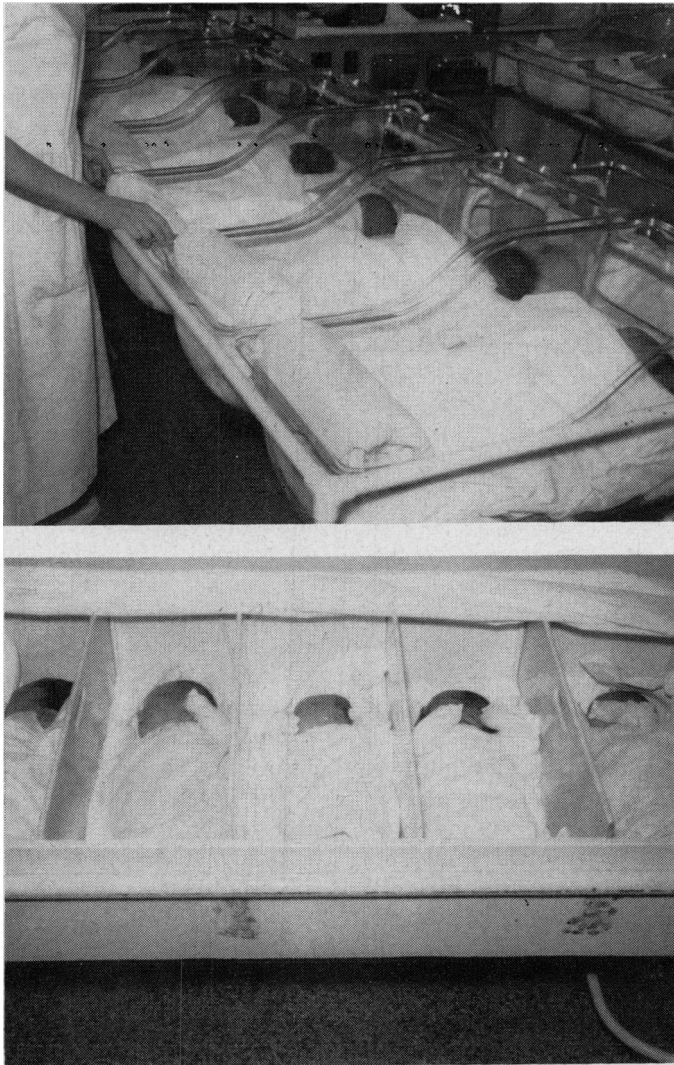
McDermott³¹ has emphasized the possible increased susceptibility of today's hospital population because of such factors as diabetes, steroid therapy, ionizing radiation, and frequent venipuncture. It is difficult to judge whether these susceptibility-increasing factors outweigh such resistance-factors as improved nutrition and reduced micro-parasitism. Likewise, the unanticipated effects of the widespread use of many antibiotics, and the unknown role of previous natural immunologic experience, complicate any judgment, whether it is increased susceptibility or increased exposure that is particularly responsible for the recent increase in hospital-derived staphylococcal disease. Until the present, however, immunization for staphylococcal disease has not proved very promising, and the continuing evolution of antibiotic-resistant strains of staphylococci limits optimism concerning the possibility that a permanently effective antibiotic will appear. Pending new technologic advances staphylococcal disease can probably best be prevented by altering the hospital environment and thereby decreasing exposure.

The hospital, to fulfill its basic purpose, must necessarily bring together infected and susceptible individuals. The inherent danger of such concentration was ably expressed by Simpson in 1869⁴³ when he wrote that "in the treatment of the sick, there is ever danger in their aggregation and safety only in their segregation . . ." Crowding of

hospital patients remains an important factor in the production of cross-infection in hospitals, especially in nurseries.^{37, 38} However, because of the propensity of those with staphylococcal infection to contaminate their environment,^{6, 12, 17, 20} and because of the great capacity of staphylococci to survive apart from human hosts,¹¹ staphylococcal disease is less dependent upon crowding for its propagation in the hospital

than are many other diseases, e.g., streptococcal infection. Bourdillon and Colebrook⁶ have demonstrated that changing of wound dressings, movement of blankets, and other activities cause rapid and substantial staphylococcal contamination of air. Duguid¹⁷ and Hare²⁰ have shown that nasal carriers contaminate their environment especially by transferring organisms with their hands to their clothing, from which staphy-

Figure 1—Crowding in Nurseries



lococcal-laden particles are subsequently liberated into the air by movement of the clothing. Colbeck¹¹ has demonstrated that patients with suppurating lesions and some carriers cause heavy contamination of many parts of the hospital environment with staphylococci, and that such contaminating organisms survive in blankets and mattresses for months and at room temperature for years. The substantial and persistent staphylococcal contamination of the hospital environment necessitates special measures to prevent indirect transmission of these parasites. Starkey⁴⁴ has provided a detailed and excellent discussion of the many factors which must be considered in a comprehensive attack upon the "sources," "depots," and "modes of conveyance" of staphylococci in hospitals. Colebrook,¹² Colbeck,¹¹ and Blowers⁴ have emphasized the importance of clean bedding, the value of using mattress covers and blankets made of heat-sterilizable cotton or synthetic fibers, and the value of oiling bedclothes to reduce dust-borne aerial contamination. Air has frequently been indicated as an important source of staphylococcal infection.^{6, 12, 28, 49} Many workers have cultured staphylococci from hospital air, and in a number of circumstances, such as in the dressing of burns and wounds, in surgery, and in greatly crowded nurseries (as shown in Figure 1), contaminated air is undoubtedly an important factor in the transmission of staphylococcal disease.^{12, 37, 21, 49} However, it is apparent from general experience and the work of Colebrook,¹² Lowbury,²⁹ and Duguid and Wallace,¹⁷ that air is only an important source or vehicle in the transmission of staphylococcal disease when highly susceptible patients, such as those listed previously, are exposed to air which has been proximally or heavily contaminated with organisms from lesions, carriers, or depots. Although it is difficult to entirely exclude droplet nuclei from the occasional trans-

mission of staphylococcal disease,⁴⁷ there is little doubt that rapid-settling, dust-borne staphylococci are of much greater importance in the aerial transmission of the disease. Special attention to air hygiene is needed in operating and dressing rooms where burns and other wounds are exposed. Colebrook¹² has found, on the basis of long experience with burn patients and intensive bacteriologic air sampling, that if proximal sources of air contamination, such as contaminated surgeon's clothing, are controlled, good air hygiene in dressing or operating rooms can be attained by positive pressure provision of 10-20 turnovers of clean air per hour. Hart²¹ has achieved marked diminution of post-operative wound infection while disinfecting operating room air with ultraviolet radiation. Special ventilation and disinfection of air can be useful adjuncts in achieving exceptionally good air hygiene in operating rooms. However, good air hygiene throughout hospitals is especially the product of good housekeeping, sterilization and oiling of blankets, clean clothing worn by clean personnel, occlusive dressings of suppurating wounds, good aseptic technic, and adequate segregation of infected and susceptible patients.

Measurement and Prevention of Staphylococcal Disease

Prevention of staphylococcal disease begins with measurement of its occurrence, and the contemporary inadequacy of such measurement jeopardizes achievement of the hospital's purpose.

Mortality statistics, as they are currently collected and analyzed, provide little knowledge of staphylococcal disease. Table 2 shows that only four of 7,837 deaths which occurred in Seattle-King County during 1956 were coded to staphylococcal disease. Yet, information derived from other sources permits an estimate that staphylococcal disease

was a major cause of certainly more than 100, and perhaps more than 200 deaths in Seattle-King County during 1956. During a four-month surveillance period in one Seattle hospital, Wysham and Kirby⁵⁰ found that "24 deaths occurred with micrococcic infections a contributing factor in each case. Twenty-one of the deaths were in the group of patients whose infections appeared during hospitalization." Examination of the 24 death certificates reveals that staphylococcal disease was mentioned on only two of the 24 certificates, one of which was coded to staphylococcal disease. Table 3 documents the sharp contrast between clinical and pathologic diagnosis and the medical certification of cause of death for 10 of the 24 deaths. Similar discrepancy between actual cause of death and death certificate information was found in an earlier study.³⁷

A combination of factors produce this gross inaccuracy of mortality statistics: (1) most physicians, including pathologists, ascertain only the anatomic site of infection at post mortem examination; rarely is a thorough attempt made to determine the etiologic agent; (2) when thorough post mortem examination is performed in an attempt to establish the etiology, the results of such studies are usually not available until after the death

certificate has been sent to the local registrar, and therefore the information is seldom entered upon the death certificate; (3) virulent staphylococcal disease is usually acquired in hospitals and, therefore, it is listed as a complication of the disease which caused the patient to seek hospital care, e.g., heart disease, burns, or cancer, and because of the current standards for the classification of deaths³⁰ such deaths are coded to the antecedent cause, that is, heart disease, burns, or cancer. There is an obvious need to encourage routine determination of the etiologic causes of death, their entry upon the death certificate, and coding of deaths to multiple causes.

In the past official health agencies have demonstrated little interest in determining the incidence and distribution of such staphylococcal diseases as osteomyelitis, mastitis, pyoderma, wound infection, and pneumonia. Now, with increased recognition of the importance of staphylococcal disease, as well as better understanding of its epidemiology, a substantial need exists for official health agencies to maintain surveillance of its occurrence. In addition to gathering reports of staphylococcal or suppurative disease occurrence from physicians, hospitals, laboratories, schools, and public health nurses, much knowledge can be gained by means of special hospital

Table 2—Mortality from Staphylococcal and Related Diseases, Seattle-King County, 1956

Information Upon Death Certificate	No. of Deaths
* Deaths coded to staphylococcal disease	4
* Staphylococcal infection mentioned	23
* Septicemia or sepsis mentioned	25
* Abscess mentioned	38
* Pneumonia or pneumonitis, etiology not stated	152
* Pneumonia mentioned with another cause	561
Total	803
Total deaths from all causes during 1956	7,837

* Each category exclusive of others.

Table 3—Index to Inadequacy of Routine Mortality Statistics (Ten Deaths with Hospital-Acquired Staphylococcal Disease a "Major Contributing Factor" to Each.*)

Case No.	Age	Data from Wysham and Kirby Study *		Data from Corresponding Death Certificates	
		Clinical and Pathological Diagnosis	Source of Culture	Medical Certification	Coded to:
1	19	"Third-degree burns, head, neck, trachea, major bronchi, and hands, micrococcic bronchopneumonia, and septicemia."	Blood	"Thermal burns"	861
4	38	"Idiopathic epilepsy; coma of unknown etiology; aspiration pneumonia with superimposed severe bilat. micrococcic bronchopneum. and lung abscess."	Left lung	"Idiopathic epilepsy"	353.3
5	87	"Myocardial infarctions, old and recent; decubitus micrococcic abscess, hips and sacrum, with probable terminal septicemia; cellulitis, left arm."	Decubitus ulcer; septic liver	"Renal infarcts; arteriosclerotic cardiovascular disease"	422.1
6	82	"Acute pyelonephritis; septicemia; abscesses of kidney and ribs; empyema." [multiple myeloma]	Rib abscess	"Multiple myeloma"	203
8	47	"Acute myeloblastic leukemia; pneumonia in left upper lobe; micrococcic septicemia; G.I. bleeding."	Blood	"Acute leukemia; myocardial infarct"	204.3
9	87	"Acute pancreatitis with suppuration; micrococcic septicemia with multiple abscesses."	Blood; urine; abdomen; rib cage	"Acute pancreatitis"	587.0
10	54	"Third degree burns of 40% of trunk and neck; micrococcic infection of burn sites."	Burn infection	"Toxemia from 2nd and 3rd degree burns."	917.0
11	59	"Multiple fractures of skull, pelvis, and legs; confluent micrococcic bronchopneumonia and septicemia."	Sputum; blood	"Lobar pneumonia, beginning lung abscesses; fract. of skull, pelvis and legs"	812.4
16	71	"Chronic pyelonephritis; massive terminal micrococcic bronchopneumonia."	Lung	"Subacute glomerulonephritis"	591
23	58	"Cerebral vascular accident; dysphagia; bilateral micrococcic bronchopneumonia of lower lobes."	Lung	"Cerebral hemorrhage; bronchopneumonia"	491

* Wysham, D. N., Kirby, W. M. M., Micrococcic (Staphylococci) Infections in a General Hospital. J.A.M.A. 164:1733-1739 (Aug. 17), 1957.

and community surveys.^{37, 38, 50} Hospital surveillance can be achieved by a systematic check of all infections and deaths occurring in hospital. Such surveillance should be guided by an "infection control committee" or a "hospital epidemiologist."¹³ Analysis of accumulated data can be facilitated by placing the data upon punch cards. Accurate and inexpensive community surveillance of neonatal and maternal morbidity, and quality control of nurseries, can be achieved by obtaining histories from mothers by telephone during their second postpartum month.^{37, 38}

At present, major reliance is placed upon measurement of morbidity and mortality for guidance of efforts aimed at preventing infection in hospitals. In the future, however, more sensitive "quality control" of the hospital environment may be achieved by routine periodic measurement of the bacterial contamination of important environmental components, such as air, blankets, floors, and people.¹²

Conclusion

There is at present a growing appreciation that a correlation does exist between staphylococcal contamination of the hospital environment and staphylococcal disease of patients and staff. No longer can staphylococci be regarded as necessarily ubiquitous in hospitals, nor discounted as "contaminants" when found in specimens from sick or dead patients. Hospital-acquired staphylococcal disease is now recognizable as a formidable contemporary cause of illness and death. Its prevention can be achieved by improved measurement of its occurrence, by forthright application of current knowledge and technics to provide a thoroughly sanitary hospital environment, and by more judicious use of improved hospitals by an informed community.

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OBSERVATIONS RELATIVE TO THE NATURE AND CONTROL OF EPIDEMIC STAPHYLOCOCCAL DISEASE

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SINCE MARCH, 1956, our study group has had opportunity to make detailed observations of nine outbreaks of staphylococcal disease among newborn infants and has been able to make limited observations of five other epidemic situations. The epidemiologic

pattern in every instance, has been remarkably similar and strongly resembles that presented by several recent publications in the British, Canadian, Australian, and American literature.¹⁻¹⁰ The outbreaks have consisted of cases of pyoderma among newborn infants